UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

ALASKA ELECTRICAL PENSION FUND, et al., Plaintiffs -against- PHARMACIA CORP., et al., Defendants.	03-1519 (AET) **DECLARATION OF TIMOTHY C. WANG, M.D.**

I, TIMOTHY CRAGIN WANG, declare, under penalty of perjury, pursuant to 28 U.S.C. § 1746, as follows: I am competent to testify to the statements made in the "Expert Report of Timothy Cragin Wang, M.D. Concerning Class Certification," dated August 30, 2006. A copy of my Expert Report is attached hereto as Exhibit 1. The statements made therein are true and correct.

Executed this 30th day of August, 2006.

Timothy Cragin Wang, M.D.

EXHIBIT 1

Alaska Electrical Pension Fund, et al. v. Pharmacia Corp., et al. No. 03-1519 (D.N.J.)

Expert Report Concerning Class Certification

Timothy Cragin Wang, M.D.

Silberberg Professor of Medicine
Columbia University College of Physicians and Surgeons

-and-

Chief of the Columbia University Medical Center
Division of Digestive and Liver Diseases

August 30, 2006

I. Background

Education and Experience

1. I am a practicing gastroenterologist and the Dorothy L. and Daniel H. Silberberg Professor of Medicine at Columbia University College of Physicians and Surgeons. I am also the Chief of the Columbia University Medical Center's Division of Digestive and Liver Diseases.

2. I am board certified in both internal medicine and gastroenterology.

3. I received my Bachelor of Arts in Chemistry *summa cum laude* in 1979 from Williams College in Williamstown, Massachusetts and my Doctor of Medicine Degree in 1983 from Columbia College of Physicians and Surgeons, New York, New York. I performed my internship and residency training in Internal Medicine from 1983 through 1986 at Barnes Hospital, Washington University School of Medicine, St. Louis, Missouri. From 1986 through 1990 I was a Research Fellow in Medicine at Harvard Medical School and a Clinical and Research Fellow in Medicine at Massachusetts General Hospital, both in Boston, Massachusetts.

4. I have been a practicing gastroenterologist for over 15 years. From 1991 to 2000 I was an Attending Physician at Massachusetts General Hospital in Boston, Massachusetts. From 2000 through 2004, I was an Attending Physician at UMass Medial Center in Worcester, Massachusetts. Since 2004, I have been an Attending Physician at New York Presbyterian Hospital. At all times during my career, a portion of my practice has been devoted to providing consultation for treatment of patients with gastrointestinal side effects related to the use of non-steroidal anti-inflammatory drugs ("NSAIDs"). I have performed endoscopic procedures on such patients and supervised residents performing endoscopic procedures. In my

current position, I presently treat patients on an in-patient basis two months per year where I see dozens of patients with NSAID-related gastrointestinal side-effects and complications.

5. I am also a professor of medicine and have been a guest lecturer and invited speaker at numerous universities and medical centers. Beginning in 1989, I was an Instructor of Medicine at Harvard Medical School, where I was promoted to Assistant Professor of Medicine in 1993 and promoted to Associate Professor of Medicine in 1998. In 2000, I joined the University of Massachusetts Medical School as a tenured Professor of Medicine where I ran the clinical, endoscopic and research programs, and also served as the Director of GI Cancer. In 2004, I joined the faculty at Columbia University College of Physicians & Surgeons where I am currently the Dorothy L. and Daniel H. Silberberg Professor of Medicine. I have also been a Visiting Professor at various universities including Yale University in New Haven, Connecticut, the University of Pennsylvania in Philadelphia, Pennsylvania, and Humboldt University in Berlin, Germany. I regularly give lectures to students, residents, fellows, and other physicians, including talks regarding NSAID-related gastrointestinal toxicity.

6. As a researcher I have conducted research in the area of gastrin biology, and continue to explore the role of gastrin peptides in diverse diseases, including peptic ulcer disease, gastric cancer and colorectal cancer. Currently, my research program encompasses six major funded projects: (1) Role of *Helicobacter pylori* in gastric cancer pathogenesis; (2) function and regulation of gastrin; (3) regulation of histidine decarboxylase gene expression; (4) function and regulation of trefoil factor 2; (5) gastric cancer stem cells; and (6) role of stromal cells in the pathogenesis of gastric and liver cancer.

7. From 1996 through 2001, I was an Associate Editor for the professional journal, *Gastroenterology*, and resumed that position on June 1, 2006. Since 2003, I have been an Associate Editor for the American Journal of Physiology (Gastrointestinal Liver Physiology). I have also reviewed for numerous professional journals, including: New England Journal of Medicine, Proceedings for the National Academy of Science, Nature Medicine, Science, Gut, Gastroenterology, American Journal of Gastroenterology, Digestive Diseases and Sciences, Digestion and the Journal of Clinical Investigation. As an editor and reviewer for many years, including the period during which COX-2 selective NSAIDs were researched and developed, I have enjoyed many opportunities to review articles that include historical reviews of NSAID development and write-ups of clinical trials and epidemiological studies relating to NSAIDs.

8. Since 1986, I have been an active member of the American Gastroenterology Association ("AGA") where I have served on numerous committees within the association, including the AGA Education Committee, the AGA Council, and the AGA Research Committee. I am currently the Chair of the AGA Future Trends Committee and a member of the AGA Leadership Cabinet. I also belong to other professional societies including the American Board of Internal Medicine ("ABIM"), the Amercian College of Physicians ("ACP"), the American Society for Clinical Investigation ("ASCI"), and the New York Society of Gastrointestinal Endoscopy ("NYSGE"). I am also a member of Phi Beta Kappa and Alpha Omega Alpha, the Honor Medical Society.

9. A copy of my *Curriculum Vitae* and a list of my publications is attached as Exhibit A.

Compensation

10. I am being compensated at my usual rate of $500 per hour in connection with this proceeding. My compensation is in no way dependent on the opinions I express or on the outcome of the case.

Prior Testimony

11. In the past four years I have submitted an expert report and given a deposition, on behalf of the plaintiffs, in connection with the following matter: *Pfizer Inc. v. Teva Pharmaceuticals USA, Inc.*, Civil Action No: 04-754 (JCL) (D.N.J.).

II. Materials Considered

12. In forming my opinions and preparing this report, I have reviewed and relied upon the materials cited and listed in this report, as well as the materials listed in Exhibit B. I have also relied on my extensive knowledge, training and experience as a gastroenterologist in forming the opinions set forth in this expert report. Reference is also made to certain documents that are attached as exhibits to the accompanying Declaration of William A. Dreier (the "Dreier Decl.").

III. Subject Matter About Which I Expect to Testify

13. I understand that this is a securities fraud action in which Plaintiffs claim that Defendants made false and misleading statements about the Celecoxib Long Term Arthritis Safety Study ("CLASS"). I further understand that Plaintiffs contend that the "truth" regarding these alleged false and misleading statements was not publicly known until the publication of a June 1, 2002 editorial by Peter Jüni, *et al.* in the British Medical Journal (the "Jüni Editorial") (Dreier Decl., Ex. 26).

14. For purposes of Defendants' opposition to the Plaintiffs' motion for class certification, I presently expect to give opinions (and, if requested, am prepared to testify) concerning (i) public knowledge of information regarding CLASS, in general, and (ii) when and the extent to which the information in the Jüni Editorial was publicly known prior to June 1, 2002.

15. I may address other matters in response to reports or other evidence offered by Plaintiffs. I reserve the right to supplement or amend my opinions in response to opinions expressed by Plaintiffs' experts, or in light of any additional evidence, testimony, discovery or other information relating to the aforementioned issues that may be provided to me after the date of this report. In addition, I expect that I may be asked to consider and testify about issues that may be raised by Plaintiffs' experts in their reports or at trial. In connection with my testimony, I may rely upon certain graphic or demonstrative exhibits that have not yet been prepared.

IV. CLASS

16. For decades before Celebrex® was approved for the U.S. market, doctors recognized that nonsteroidal anti-inflammatory drugs (NSAIDs) are associated with gastrointestinal side effects, including peptic ulcers, ulcer complications and tolerability issues (such as nausea, abdominal pain and dyspepsia). The medical community, therefore, realized that there was a need for new NSAIDs with improved gastrointestinal safety and tolerability compared to the NSAIDs available to their patients.

17. With that background in mind, Celebrex® was developed and subsequently approved for marketing in the United States in December 1998.[1] Numerous endoscopic studies, outcome studies and epidemiologic studies show that treatment with Celebrex® is associated with fewer gastrointestinal side effects and has better GI tolerability compared to "traditional" or "non-selective" NSAIDs on the market in the United States.[2] These studies, as a whole, lead to the conclusion that Celebrex® has a gastrointestinal safety benefit and that reduced risk in comparison to other NSAIDs is the driving force behind doctors' decisions to prescribe Celebrex® for patients in need of NSAID therapy.

18. Based on the acknowledged risk for upper gastrointestinal side effects associated with NSAID use, the FDA has required that approved labeling for prescription NSAIDs include a standard paragraph explaining "that upper GI ulcers, gross bleeding or perforation, caused by NSAIDs, appear to occur in approximately 1% of patients treated for 3-6 months, and in about 2-4% of patients treated for one year" (the "Standard GI Warning").[3]

19. Notwithstanding the evidence demonstrating the superior GI safety profile of Celebrex, the FDA required that the label for Celebrex also include the Standard GI Warning.

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[1] On December 31, 1998, the United States Food and Drug Administration ("FDA") approved Celebrex® -- the first Cox-2 selective NSAID to be approved in the United States -- for treatment of patients with rheumatoid arthritis and osteoarthritis. The FDA found that Celebrex® was an effective arthritis treatment in placebo and active-controlled clinical trials and demonstrated a substantially lower risk of ulcers detected by endoscopy. *See* "FDA Talk Paper" *FDA Approves Celebrex for Arthritis* (Dec. 31, 1998). Subsequently, the FDA approved Celebrex® for additional indications.

[2] The FDA has categorized Celebrex®, Vioxx® and Bextra® as Cox-2 selective NSAIDS and other drugs, such as ibuprofen, etodolac, meloxicam, nabemutone and diclofenac as non-selective NSAIDs. I use the FDA's nomenclature for this report.

[3] *See* Harold E. Paulus, *FDA Arthritis Advisory Committee Meeting: Serious Gastrointestinal Toxicity of Nonsteroidal Antiiflammatory Drugs; Drug -Containing Renal and Bilary Stones; Diclofenac and Carprofen Approved*, Vol. 31, No. 11, Arthritis and Rheumatism 1450-51 (1988); *see also Medical Officer Review* by James Witter, M.D., Ph.D. ("Witter Review") at 5-6 (Dreier Decl., Ex. 11).

20. The CLASS study was commissioned to compare, among other things, the GI safety profile of Celebrex® to that of traditional NSAIDs in order to support Pharmacia's petition to the FDA for approval of a revised label.

21. The protocol for CLASS specified that Celebrex® would be compared to two commonly used NSAIDs, ibuprofen and diclofenac. The determination of the relative GI safety of Celebrex® versus these other NSAIDs would be made primarily by reference to the incidence of "ulcer complications" – defined, for purposes of the Study, as upper gastrointestinal bleeding, perforation or gastric outlet obstruction in patients with osteoarthritis or rheumatoid arthritis. The incidence of "ulcer complications" was designated as the "primary endpoint," or, objective, of CLASS. The incidence of a secondary endpoint of "symptomatic ulcers" – defined, for purposes of the Study, as ulcers identified based on upper gastrointestinal symptoms, such as abdominal pain, dyspepsia, nausea, diarrhea or vomiting – was included in the protocol as part of the evaluation of general GI safety. The Study was designed to include approximately eight thousand patients. Approximately four thousand patients would be given Celebrex®, two thousand ibuprofen and two thousand diclofenac. The minimum planned study participation was 6 months.

22. The results of CLASS were reported in an article published in the Journal of the American Medical Association on September 13, 2000, entitled *Gastrointestinal Toxicity With Celecoxib vs Nonsteroidal Anti-inflammatory Drugs for Osteoarthritis and Rheumatoid Arthritis - The CLASS Study: A Randomized Controlled Trial* (the "JAMA Article") (Dreier Decl., Ex. 1).

23. The JAMA Article reported that on the primary endpoint, Celebrex failed to demonstrate that there were statistically significantly fewer ulcer complications in the Celebrex treatment group than in the comparator treatment groups. However, the JAMA Article reported that according to various other measures, Celebrex demonstrated superiority over the comparator NSAIDs. Specifically, based on the first six months of data from the study, there were statistically significantly fewer complicated and symptomatic ulcers as compared to the pooled NSAID comparators. Dreier Decl., Ex. 1.

24. I understand that Plaintiffs' complaint in this action criticizes the JAMA Article. Specifically, Plaintiffs contend that Defendants misrepresented the results of the CLASS Study (1) by using 6 months worth of data and not reporting on the longer-term data that was available; (2) by using a combined endpoint consisting of symptomatic ulcers (a "secondary endpoint") in addition to ulcer complications (the "primary endpoint"); (3) by not following the prespecified plan for statistical analysis of the primary endpoint which called for (i) an analysis of the Celebrex v. ibuprofen and diclofenac, combined, and then (ii) an analysis of Celebrex v. ibuprofen and of Celebrex v. diclofenac, individually; and (4) because "[a]nalyzing the CLASS Study data pursuant to the original protocol, meaning 12 and 15 months of data compared head-to-head and in combination for ulcer-related complications, Celebrex does not offer greater GI safety than traditional NSAIDs." Complt. ¶ 46.

25. I disagree with such criticism and it is my opinion that the analysis offered in the JAMA Article is not misleading, and represents a valid interpretation and presentation of the CLASS data.

26. Nevertheless, for the purposes of this report, I have been asked to assume, for the sake of argument, that the results of CLASS, as presented in the JAMA Article, were misrepresented, and to offer my opinion as to when the "truth" regarding such alleged misrepresentations was publicly revealed. My opinion is that the "truth" was publicly revealed no later than February 6, 2001, when the full data from CLASS was made publicly available on the FDA website.

V. The "sNDA" And FDA Arthritis Advisory Committee

27. Pharmacia's application to the FDA to remove the Standard GI Warning is technically known as a "Supplemental New Drug Application" or "sNDA." The Arthritis Advisory Committee, a committee of outside experts appointed by FDA, was tasked with evaluating the results of CLASS and making a recommendation to the FDA regarding the requested label change. In advance of the Advisory Committee meeting, which was held February 7, 2001, Pharmacia submitted a "briefing document," setting forth its analysis of the results of CLASS (the "Briefing Document") (Dreier Decl. Ex. 9).[4]

28. The Briefing Document presented the CLASS data in two ways: (1) using six months of data (as in the JAMA Article) and (2) using data from the entire study period.

29. As a basis for using six months of data, the Briefing Document explained that data after six months was suspect and not statistically meaningful because of disproportionate withdrawal of patients in the ibuprofen treatment arm generally and in the diclofenac treatment arm for GI adverse events specifically:

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[4] Technically, the Briefing Document was submitted by G.D. Searle & Co. ("Searle") which I understand is affiliated with Pharmacia. Accordingly, for purposes of this affidavit any references to Pharmacia include Searle.

> The GI safety data presented are for the six-month treatment timepoint based on the analysis of risk factors prespecified in the protocol. In brief, a disproportionate withdrawal of patients at high risk of an ulcer complication from the entire study was observed after six months (depletion of susceptibles). Additionally, a significantly greater withdrawal of patients on diclofenac for GI intolerance occurred during the initial six months of the study. The withdrawal of patients for GI intolerance prematurely removed a group at high risk for ulcer complications and symptomatic ulcers from the diclofenac treatment arm (informative censoring).

Briefing Document (Dreier Decl., Ex. 9) at 28.

30. FDA Medical Officers and an FDA Statistician submitted detailed reviews of the CLASS data to the Arthritis Advisory Committee for consideration. Specifically, Dr. James Witter, an FDA Medical Officer, submitted a "Medical Officer Review" (Dreier Decl., Ex. 11), Dr. Lawrence Goldkind, an FDA Medical Officer, submitted a "Medical Officer's Gastroenterology Advisory Committee Briefing Document" (the "Goldkind Review") (Dreier Decl., Ex. 10) and Dr. Hong Laura Lu, an FDA Statistician, submitted a "Statistical Reviewer Briefing Document for the Advisory Committee" (the "Lu Review") (Dreier Decl., Ex. 12) (collectively, the "FDA Staff Review Documents"). The Briefing Document and the FDA Staff Review Documents were made publicly available on February 6, 2001, when they were posted to the FDA website. They remain available there today.[5]

31. On February 7, 2001, the FDA Arthritis Advisory Committee held a public hearing at which representatives of Pharmacia and FDA staff (Medical Officers) made presentations to the committee.

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[5] *See* http://www.fda.gov/ohrms/dockets/ac/01/briefing/3677b1.htm.

32. Each of the alleged misrepresentations set forth in Paragraph 24, above (summarizing ¶ 46 of the Complaint), was discussed in the Briefing Document and the FDA Staff Review Documents, and during the Arthritis Advisory Committee public hearing.

The Duration Of CLASS

33. Plaintiffs contend that Defendants misrepresented the duration of the CLASS Study because "[t]he CLASS study trial did not last six months as stated. Instead, the Celebrex versus ibuprofen trial lasted 15 months and the Celebrex versus diclofenac trial lasted 12 months." Complt. ¶ 46. This information was publicly known as early as April 17, 2000.

34. Pharmacia's initial press release, issued on April 17, 2000, regarding the CLASS results, stated that the CLASS Study was "an approximately 13 month" study. Dreier Decl., Ex. 2 at 2. This was echoed in various publications, such as the April 24, 2000 edition of the "Pink Sheet," an industry newsletter covering the Pharmaceuticals industry (Dreier Decl., Ex. 7 at 1-2), and two April 17, 2000 articles that were reported on the Dow Jones newswire. *See* Dreier Decl., Exs. 4-5.

35. The Briefing Document and the FDA Staff Review Documents, made public on February 6, 2001, also clearly identified the duration of the CLASS Study and the time periods relevant to Pharmacia's interpretation of the data. *See* Briefing Document (Deier Decl., Ex. 9) at 18, 28, 45; Goldkind Review (Dreier Decl., Ex. 10) at 12, 14-16, 23-24; Witter Review (Dreier Decl., Ex. 11) at 8, 48; Lu Review (Dreier Decl., Ex. 12) at 1, 5, 8.

36. In addition, comments made by Pharmacia representatives during the Arthritis Advisory Committee public hearing also made clear that the CLASS Study lasted longer than six months. *See* Dreier Decl., Ex. 17 (excerpts of hearing transcripts).

Alleged Expansion Of The Primary Endpoint

37. Plaintiffs contend that the Defendants misrepresented the results of CLASS because "after the trials were complete, defendants added symptomatic ulcers to the comparison criteria in order to improve Celebrex's relative performance." Compl. ¶ 46(c).

38. It was well understood long before the JAMA Article was published, however, that Celebrex failed to meet the primary endpoint of CLASS. For example, in an April 18, 2000 report entitled *Positive Results of Celebrex CLASS Trial Released*, Morgan Stanley Dean Witter reported that, "Celebrex did not reach statistically significant superiority on the primary endpoint of ulcer complications." Dreier Decl., Ex. 33 at 2. This information was similarly reported in the April 24, 2000 edition of the Pink Sheet. Dreier Decl., Ex. 7 at 2.

39. Moreover, the JAMA Article specifically disclosed that Celebrex failed to meet the primary endpoint. *See* JAMA Article (Dreier Decl., Ex. 1) at 1254 ("the rate for ulcer complications did not differ"). Further, in an editorial commenting on the results of CLASS, published in JAMA contemporaneously with the JAMA Article, the authors noted that "even though the combined incidence of symptomatic ulcers . . . associated with celecoxib was significantly lower than with the comparator drugs, careful examination of the data shows that the rate of ulcer complications alone, the primary end point of the study, was not." David R. Lichtenstein, M.D. and M. Michael Wolfe, M.D., *COX-2-Selective NSAIDs, New and Improved?*, Vol. 284, No. 10 J. of Am. Med. Ass'n 1297, 1297-99 (Sept. 13, 2000) (Dreier Decl., Ex. 32).

40. The FDA Staff Review Documents also discussed that Celebrex failed to meet the primary endpoint and that the use of a combined endpoint of ulcer complications and

symptomatic ulcers was a post-hoc analysis, with symptomatic ulcers a prespecified secondary endpoint. *See* Goldkind Review (Dreier Decl., Ex. 10) at 21, 41, 52; Lu Review (Dreier Decl., Ex. 12) at 9, 6.

Alleged Failure To Follow The Prespecified Statistical Analysis

41. Plaintiffs allege that Defendants misrepresented the results of CLASS by not following the prespecified plan for statistical analysis of the results which called for (i) an analysis of the Celebrex v. ibuprofen and diclofenac, combined, and then (ii) an analysis of Celebrex v. ibuprofen and of Celebrex v. diclofenac, individually. The protocol explicitly specified that Celebrex would only be found superior . . . if both comparisons, [Celebrex vs. diclofenac and Celebrex vs. ibuprofen], were statistically significant and in favor of Celebrex." Cmplt. ¶ 46.

42. Plaintiffs' criticism, however, was publicly discussed in the Goldkind Review, published on February 6, 2001. *See* Dreier Decl, Ex. 10 at 7. Moreover, the protocol called for the primary endpoint (1) to be analyzed at the combined NSAID level and (2) only if the difference were statistically significant to proceed to specific comparisons of ibuprofen and diclofenac. As fully disclosed, the primary endpoint was not met and therefore the protocol did not call for comparisons with specific comparators. The protocol did not prescribe a specific methodology for conducting the secondary analyses.

The Alleged Absence Of Any Safety Advantage According To The CLASS Protocol

43. Plaintiffs contend that Defendants misrepresented the results of CLASS because the JAMA Article did not disclose that if the data is analyzed "pursuant to the original protocol, meaning 12 and 15 months of data compared head-to-head and in combination for

ulcer-related complications, Celebrex does not offer greater GI safety than traditional NSAIDs." Cmplt. ¶ 46. Plaintiffs' criticism, as well, was publicly known, as it was discussed in the Witter Review, published on February 6, 2001. *See* Dreier Decl., Ex. 11 at 48, 82.

44. In addition, comments made by Pharmacia representatives and FDA representatives during the Arthritis Advisory Committee public hearing also made clear that, according to the protocol, Celebrex did not show any statistically significant difference over the comparators. *See* Dreier Decl., Ex. 17.

45. In my opinion, the posting of the Briefing Document and the FDA Staff Review Documents on the FDA website, as well as the public statements made during the FDA Arthritis Advisory Committee hearing, publicly stated each of Defendants' alleged misrepresentations regarding the results of CLASS. Indeed, most of Plaintiffs' allegations were publicly known as early as April 2000. In addition to the evidence discussed above, my opinion is confirmed by the fact that news outlets and securities analysts published reports that reviewed and analyzed the materials posted on the FDA website and, accordingly, discussed the very facts that are alleged to have been misrepresented and/or not disclosed. *See, e.g.*, February 7, 2001 J.P. Morgan Securities analyst report entitled *FDA Review of Celebrex More Negative Than Expected – Panel Could Be Controversial* (Dreier Decl., Ex. 15); Brian Reid, *Pharmacia Hasn't Shown Celebrex Safety Benefit, FDA Review Says*, Bloomberg News, Feb. 6, 2001 (Dreier Decl., Ex. 16).

46. Moreover, at the conclusion of the Arthritis Advisory Committee hearing, the Committee publicly disagreed with Pharmacia's conclusions that the CLASS Study demonstrated the superior GI safety profile of Celebrex. The Committee concluded that CLASS

did not demonstrate the superiority of Celebrex over NSAIDs because Celebrex had not met the primary endpoint and recommended to the FDA that the gastrointestinal warning not be removed from the Celebrex label. The Arthritis Advisory Committee's conclusion was widely reported in securities analyst reports and the general press. *See* Dreier Decl., Exs. 19-20. It should be noted, however, that the FDA ultimately disagreed with the conclusions and recommendation of the Advisory Committee and other critics by approving a label change based on a nine-month analysis of the CLASS Study and a combined endpoint of symptomatic ulcers and ulcer complication. *See* "FDA Talk Paper" entitled *Labeling Changes for Arthritis Drug Celebrex* (June 7, 2002) (Dreier Decl., Ex. 18).

47. I understand that notwithstanding all of the above, Plaintiffs contend that the alleged fraud was "perpetuated" when Defendants "caused three of the authors of the CLASS study . . . to publish a letter in the November 21, 2001 issue of JAMA," which gave allegedly false and misleading explanations to justify the six month analysis offered in the JAMA Article. Complt. ¶ 67 (the "November 21 JAMA Letter") (Dreier Decl., Ex. 21). Thus, Plaintiffs contend that the "truth" about Defendants' alleged misrepresentations regarding the results of the CLASS Study was not revealed until June 1, 2002, when the Jüni Editorial "exposed" the explanations set forth in the November 21 JAMA Letter as "lies." Complt. ¶ 69.

48. In my opinion, Plaintiffs are wrong because the so-called "truth" articulated by Plaintiffs about the results of CLASS had already been publicly known no later than February 6, 2001. Specifically, the points made in the Jüni Editorial were the same points made in the FDA Staff Review Documents (posted on the FDA website on February 6, 2001) and during the Arthritis Advisory Committee public meeting on February 7, 2001. The allegedly false and misleading explanations offered in the November 21 JAMA Letter, were the same

explanations that were given in the Briefing Document, which the FDA Staff Review Documents critiqued in public postings on February 6, 2001. Thus, Defendants' explanations of the use of six-month data and a combined endpoint, and the FDA Staff Review Documents' critique thereof – the same as Jüni's – were publicly known no later than February 6, 2001.

49. According to Plaintiffs, the Jüni Editorial[6] "revealed these "excuses [for the 6-month analysis] as nothing more than lies" Complt. ¶ 69. Plaintiffs assert that the Jüni Editorial revealed that, contrary to Defendants' representations, there was no "differential drop-out rate," between treatment groups and that there was no basis for the contention that there was "depletion of susceptibles" or informative censoring, because there is no evidence to support the notion that symptomatic ulcers lead to ulcer complications. As discussed above, however, these same points were explicitly discussed in the FDA Staff Review Documents, and, thus made public no later than February 6, 2001. *See* Goldkind Review (Dreier Decl., Ex. 10) at 21-22, 33; Lu Review (Dreier Decl., Ex. 12) at 8-9.

50. Thus, the Jüni Editorial revealed no information that had not already been discussed in the FDA Staff Review Documents, if not earlier. In fact, the conclusions of the authors of the Jüni Editorial are acknowledged to be based upon the authors' review of the FDA Staff Review Documents. As authority for the very proposition that Plaintiffs' claim "finally revealed" the "truth," (Complt. ¶ 71), the authors of the Jüni Editorial cite to the FDA Staff Review Documents, which, according to that citation, they reviewed on December 10, 2001. *See* Jüni Editorial (Dreier Decl., Ex. 26) at 1288 nn.6, 7, 8.

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[6] *See* Dreier Decl., Ex. 26.

Dated: August 30, 2006

Timothy Cragin Wang, M.D.

EXHIBIT A

CURRICULUM VITAE (UPD. 3/08/06)

Name: **Timothy Cragin Wang, M.D.**

Address: 455 Central Park West, Apartment #11C
New York, NY 10025

Date of Birth: March 27, 1957

Place of Birth: Allentown, Pennsylvania

Education:

1975:		John Burroughs Preparatory School, Ladue, Missouri
1979	B.A.	Williams College, Williamstown, Massachusetts
1983	M.D.	Columbia College of Physicians and Surgeons, New York, New York

Postdoctoral Training:

Internships and Residencies:

1983-1986	Intern and Resident in Internal Medicine, Barnes Hospital, Washington University School of Medicine, St. Louis, Missouri

Research Fellowships:

1986-1989	Research Fellow in Medicine, Harvard Medical School
1986-1989	Clinical and Research Fellow in Medicine, Massachusetts General Hospital, Boston, Massachusetts

Licensure and Certification:

1987	Massachusetts License Registration No. 55632
2004	New York License No. 233070-1

Academic Appointments:

1989	Instructor of Medicine, Harvard Medical School
1993	Assistant Professor of Medicine, Harvard Medical School
1998	Associate Professor of Medicine. Harvard Medical School
1999	Professor of Medicine (with tenure), University of Massachusetts Medical School
2004	Assistant Adjunct Professor of Medicine, Columbia University College of Physicians & Surgeons
2005	Dorothy L. and Daniel H. Silberberg Professor of Medicine Columbia University College of Physicians & Surgeons

Hospital Appointments:

1989-1991	Clinical Assistant in Medicine, Massachusetts General Hospital
1991	Assistant in Medicine, Massachusetts General Hospital
1994	Assistant Physician, Massachusetts General Hospital
1998	Associate Physician, Massachusetts General Hospital
	Associate Division Chief, Massachusetts General Hospital
2000	Chief, Gastroenterology Division,
	Director of GI Cancer,
	University of Massachusetts Medical School
2004	Chief, Digestive and Liver Diseases
	Director of GI Cancer
	Columbia University Medical Center

Awards and Honors:

1979	B.A., summa cum laude
1983	M.D. (AOA)
1988	AGA Senior Research Fellow Award
1993	AGA Funderberg Gastric Cancer Award
1998	Election to American Society of Clinical Investigation (ASCI)
1999	Steven Krane Lectureship for Outstanding Young Investigator in the MGH Department of Medicine
2000	Viktor Mutt Medal in Gut Hormone Research
2001	Gladys Smith Martin Chair in Gastrointestinal Cancer
2004	Election to Association of American Physicians (AAP)
2005	Silberberg Chair in Medicine, Columbia P&S

Major Committee Assignments:

Hospital	
1993	Committee on Research, MGH
1993	Subcommittee for Research Animal Care/Studies, MGH
1998	Steering Committee, Gastrointestinal Unit, MGH
1999	Academic Governing Council, Department of Medicine, MGH
2000	Appointments and Promotions Committee, UMass Med School
2000	Member of Cancer Center Search Committee, UMass Medical School
2002	Board Member of Cancer Center Programs Leaders at UMass Medical School
2003	Member of Endoscopy Working Group, UMass Medical School
2003	Member of Pediatric GI Search Committee, UMass Medical School
2004	Department of Medicine Executive Council, Columbia P&S
2005	Member of Radiation Oncology Search Committee, Columbia P&S

National Committee Assignments:

1996-1998	AGA By-Laws Committee
1999-2002	AGA Education Committee
2001-2005	AGA Council, Hormones & Receptors Section
2002-2004	AGA Research Committee
2003	AGA Nominating Committee
2003-2004	Director, AGA 2004 Spring Postgraduate Course (SPGC)
2003-2004	AGA Strategic Planning Committee
2003-5	AGA GRG Committee
2004-2005	AGA Research Policy Committee
2005-2008	Chair, AGA Future Trends Committee

Memberships in Professional Societies:

1979	Phi Beta Kappa
1983	Alpha Omega Alpha
1983-	American Medical Association
1986-	American Gastroenterology Association (AGA)
1986-	American Board of Internal Medicine (ABIM)
1986-	American College of Physicians (ACP)
1998	American Society for Clinical Investigation (ASCI)
2001	American Physiological Society (APS)
2002	American Society for Biochemistry and Molecular Biology (ASBMB)
2003	Crohn's & Colitis Foundation of America (CCFA)
2004	Association of American Physicians (AAP)
2005	New York Society of Gastrointestinal Endoscopy (NYSGE)

Editorial Experience:

1996-2001	Associate Editor, Gastroenterology
2003-2007	Associate Editor, Am. J. Physiol. (*Gastrointest Liver Phys*)
2006-2010	Associate Editor, Gastroenterology

Journals Reviewed For:

1. Nature Medicine
2. Science
3. New England Journal of Medicine
4. Proceedings for the National Academy of Science
5. EMBO J
6. Journal of Clinical Investigation
7. Endocrinology
8. Cancer Research
9. FEBS Letters
10. Biochemistry Journal
11. FASEB Journal
12. Hepatology

13. Gut
14. Gastroenterology
15. American Journal of Physiology
16. Journal of Cell Physiology
17. Regulatory Peptides
18. American Journal of Gastroenterology
19. Cell Growth & Differentiation
20. Digestive Diseases and Sciences
21. Digestion

Major Research Interests:

1. Regulation of histidine decarboxylase gene expression
2. Role of gastrin in growth and colon cancer
3. The role of cyclin D1 in oncogenesis
4. Mouse models Helicobacter pylori and gastric cancer
5. Function and regulation of trefoil factor 2 (TFF2/SP)
6. Regulate of innate immunity
7. Stem cells and cancer

RESEARCH FUNDING:

CURRENT FUNDING:

NIH RO1CA120979
Funding agency: NIH NCI
Title: Stem cells and gastric cancer
Principal investigator: Timothy C. Wang
Dates of Award: 3/06 to 2/11

NIH RO1 DK 48077
Funding agency: NIH NIDDK
Title: The regulation of histidine decarboxylase gene expression
Principal investigator: Timothy C. Wang
Dates of Award: 6/04 to 6/09

NIH RO1 DK52778
Funding agency: NIH NIDDK
Title: Function and regulation of gastrin
Principal investigator: Timothy C. Wang
Dates of Award: 03/01/04 to 02/28/09

NIH RO1 CA93405
Funding agency: NIH NCI
Title: Mouse models of gastric cancer
Principal Investigator: Timothy C. Wang

Dates of Award: 4/1/01 to 3/31/06

NIH RO1 DK58889-01
Funding agency: NIH NIDDK
Title: Function and regulation of spasmolytic polypeptide/TFF2
Principal Investigator: Timothy C. Wang
Dates of Award: 3/1/01 to 2/28/06

NIH RO1 AI51405-01
Funding agency: NIH NAID
Title: Heath shock proteins and *Helicobacter pylori* pathogenesis
Principal Investigator: Evelyn Kurt-Jones; Co-PI: Timothy C. Wang
Dates of Award: 4/01/02 – 3/31/07

RECENT PAST FUNDING: Not included.

POST-DOCTORAL FELLOWS PAST:

NAME	TRAINING PERIOD	RESEARCH PROJECT	CURRENT POSITION
Michael Hoecker	1993-1996	Role of Gastrin in Chromogranin a Regulation	Associate Professor of Medicine, Charite Med School, Humboldt University
Zhensheng Zhang	1993-1996	Human HDC Promoter	Assistant Professor of Medicine Memorial Medical School, NIH
Ted Koh	1994-1997	Role of Gastrin in Gastro-Intestinal Development	Assistant Professor of Medicine Syracuse Hospital, New York
Robert Henihan	1996-1998	Regulation of the HDC Promoter by somatostatin	Wilmington Gastroenterology Associates, North Carolina
Raktima Raychowdhury	1996-2000	Gastrin Regulation of the HB-EGF Gene	Research Associate Professor of Medicine, Harvard University
John Fleming	1997-2003	Expression and Regulation of Histidine Decarboxylase	Research Assistant Professor, Institute of Molecular Medicine Lisoboa, Portugal
Woo Kyu Jean	1997-1998	Regulation of TFF2	Assistant Professor of Medicine Samsung University, Seoul, Korea
Clemens Bulitta	1998-2000	Regulation of gastrin and TFF2	Siemens Medical Solutions Health Services GmbH Erlangen, Germany
Rocchina Colucci	1997-1999	Regulation of HDC promoter	Research Assistant Professor

			Faculty of Medicine, Pisa Univ
James Farrell	1998-2001	Generation and Characterization of TFF2 Deficient Mouse	Assistant Professor of Medicine UCLA School of Medicine
John McLaughlin MD, PhD	1998-1999	Gastrin Regulation of the HB-EGF Gene	Assistant Professor of Medicine Manchester University, England
Abhijit Chaklader PhD	2000-2002	Molecular Characterization of Human Rotaviruses Isolated from Clinical Cases	Staff Scientist, Harvard Medical School, UMass Medical School
Chung-Wei Lee MD	2000-2001	Regulation of Mouse TFF2 Promoter Activity	PhD. Student, Massachusetts Institute of Technology (MIT)
Guanglin Cui MD	2001-2003	The Role of Cytokines in Gastritis	University of Tromso Tromso, Norway
SeonHee Lim MD. PhD.	2002-2003	Regulation of TFF2 Gene Expression	Assistant Professor of Medicine Kang Nam Hospital, Seoul, Korea
Alfred Chi PhD	2001-2004	Regulation of TFF2	Founder of biotech company
Shi Lei PhD	2001-2004	Regulation Of Gastrin Gene Expression	Postdoc with Dr. Steve Reppert Neurobiology, UMass

CURRENT POST-DOCS

Wandong Ai, PhD	2002-Present	Regulation of HDC Promoter Activity	Associate Research Scientist Columbia P&S
Shigeo Takaishi MD, PhD	2002-Present	Mouse Model of Gastric Cancer	Associate Research Scientist Columbia P&S
Alexander Dubeykovskiy, PhD	2003-Present	Progastrin and Cancer	Associate Research Scientist Columbia P&S
Zina Dubeykovskaya, PhD	2004-Present	Trefoil Family Factor 2	Post-doctoral Research Scientist Columbia P&S
Shuiping Tu, MD, Ph.D	2004-Present	Mouse Models of Gastric Cancer	Post-doctoral Research Scientist Columbia P&S
Iva Smirnova, MD, PhD	2004-present	Gastric Progenitor Cells	Post-doctoral Research Scientist Columbia P&S
Tomoyuki Okumura MD, PhD	2005-Present	Stem Cells and Cancer	Post-doctoral Research Scientist Columbia P&S
Frederic Marrache MD, PhD	2005-Present	Models of Pancreatic Cancer	Post-doctoral Research Scientist Columbia P&S
Sheng-Wen Wang MD, PhD	2005-Present	Inflammation and cancer	Post-doctoral Research Scientist Columbia P&S

Principal Clinical and Hospital Service Responsibilities:

1991-	Attending Physician, Medical Service, Massachusetts General Hospital
2000-	Attending Physician, UMass Medical Center
2004-	Attending Physician, New York Presbyterian Hospital

Major Administrative Responsibilities:

1992:	Director of GI Unit Research Journal Club
1993:	Co-Director, Course on Techniques in Molecular and Cellular Biology
1995:	Director of Transgenic Core Laboratory, CSIBD
1996:	Director of GI Unit Research Seminar Series
1998:	Director of Partner's dyspepsia disease management program
1998:	Associate Chief, Gastrointestinal Unit, MGH
2000:	Chief, Gastrointestinal Division, UMass Medical
2004:	Chief, Gastroenterology Division, Columbia University Medical School

Teaching Experience:

1991-2000	Visit on the Gastrointestinal Service
1992-2000	Visit on the Bigelow Medical Service
1993-2000	Co-director of the IBD Center Molecular Biology Course
1993-2004	Lecturer at Harvard Postgraduate course in Gastroenterology, Infectious Diseases, and Surgery
1998-2000	Lecturer in CME program: AMIL Physicians from Brazil
2000-2004	Attending on the UMass Medicine and GI services
2000-2004	Co-director of GI Pathophysiology course, UMass Medical School
2001-2005	Thesis committee Tox/Path for M.I.T.
2002, 2004, 2005	AGA, Academic Skills Workshop

Invited Speaker:

1991	University of Pennsylvania
1992	University of Michigan
1993	Boston University
	Peptide Growth Factor Conference, Vail, Colorado
1994	Visiting Lecturer, CURE, UCLA
1995	Tufts University/New England Medical Center
	Massachusetts Institute of Technology
1996	GI Cancers: Biology and Genetics, Reston, VA
1997	Gastrin Conference, Liverpool, UK
	MIT, Division of Toxicology
1998	Visiting Lecturer, Univ. of Michigan
	Visiting Lecturer, UTMB
	Visiting Lecturer, Medical College of Georgia
	K-Club, DDW, New Orleans

	Yamanouchi Symposium, Tokyo Japan
	Visiting Professor, University of Cincinnati
1999	Keystone Symposium on GI Cancer
	Chugai Symposium, DDW, Orlando
	Visiting Professor, National University, Taiwan
	Visiting Lecturer, Mount Sinai Medical Center
2000	Visiting Lecturer, University of Massachusetts
	Visiting Lecturer, Brigham and Woman's Hospital
	Visiting Lecturer, Emory University
	Visiting Lecturer, Massachusetts Institute of Technology
	Visiting Lecturer, Merck Corporation, Pennsylvania
	Visiting Professor, Humboldt University, Berlin, Germany
	Victor Mutt Lectureship, Cairns, Australia
	External Scientific Advisory Board, Washington Univ. Nutrition Center
2001	Visiting Professor, University of Pennsylvania
	Visiting Professor, University of Connecticut
	Visiting Professor, Yale University
	Invited Speaker, Worcester Medical Center/St. Vincent's Hospital
	Invited Speaker, APDW, Sydney, Australia
	Invited Speaker, Conference on gastric cancer, Astra-Zeneca, Waltham, MA
	Invited Speaker, Keio and Kyote Universities, Japan
	Invited Speaker, FASEB, Kalispell, Montana, July 2001
	Invited Speaker, FASEB, Ottawa, Quebec, Canada, October 2001
	Invited Speaker, University of Toronto
	Invited Speaker, 3rd International Conference on Trefoils April
2002	Invited Speaker, Pacific Basin Group on Gastroduodenal Disorders Meeting
	Invited Speaker, University of Pennsylvania Postgraduate Course
	Invited Speaker, Yale University School of Medicine
2003	Invited Speaker, Beth Israel Deaconess Hospital, Boston
	Salmon Visiting Professor of Medicine: Vanderbilt University
	Invited Speaker: "Biology of gastrointestinal bleeding", Yale University
	Invited Speaker: "*Helicobacter pylori* and gastric cancer", Columbia College of Physicians and Surgeons, NY.
	Invited Speaker: "*Helicobacter pylori* and gastric cancer", Duke University Medical Center
	Invited Speaker: *Helicobacter* and gastric cancer, Astra-Zenneca International Consensus Conference, Stockholm, Sweden.
	Grand Rounds speaker: *H. pylori*: Update 2003, St. Vincent's Medical Center, Worcester, MA.

Grand Rounds speaker: *H. pylori*: Update 2003, Holy Family Hospital, Methuen, MA.
Invited speaker: Infection and inflammation: the origins of epithelial cancers, Beth Israel Deaconess Hospital, Boston, MA

2004 Invited Speaker: Massachusetts General Hospital, "Trefoil factor 2: non-epithelia expression and the origin of epithelial cancers"
Invited Speaker: UCLA, VA Healthcare System, CURE "Helicobacter and Gastric Cancer"
Invited Speaker, Dana Farber Cancer Center, "Helicobacter, Inflammation and Gastric Cancer"
Invited Speaker at Massachusetts General Hospital, GI Grand Rounds "H pylori and Gastric Cancer" in the Isselbacher Library
Invited Speaker: 15th International Symposium of Regulatory Peptides, "Non-processed gastrins: effects, mechanisms of action and pathophysiological relevance", Toulouse, France
Pediatric GI Grand Rounds, "*Helicobacter* and gastric cancer", Columbia University Medical Center
Invited lecture: Cornell Medical School, GI Grand Rounds, "*Helicobacter pylori* and gastric cancer." Columbia College of P&S, Medical Grand Rounds, "*H. pylori* and gastric cancer."
Invited speaker, 4th International Conference on Trefoil factors, Strasbourg, France. "Trefoil factor-2 (TFF2): non-epithelial expression and possible role in immune regulation."

2005

January Lecture to GI fellows at Columbia on "Peptic Ulcer Disease"
Invited lecture: GI Grand Rounds, "*Helicobacter* and gastric cancer" St. Luke's/Roosevelt Hospital, New York, NY

March Invited speaker: Stanford University Microbiology, Molecular Oncology Research Seminar, "*Helicobacter* pylori and gastric cancer: anew paradigm for epithelial cancer", Palo Alto, CA
Invited lecture: GI Grand Rounds, "Helicobacter and gastric cancer: a new paradigm for inflammation mediated neoplasia", Stanford University, Palo Alto, CA
Invited speaker: Genentec Molecular Oncology Seminar, "Helicobacter pylori and gastric cancer: a new paradigm for cancer stem cells", South San Francisco, CA
Invited speaker: Washington Hospital Center, Cardiovascular Revascularization Therapies (CRT) 2005 Conference, "Studies suggesting that stem cell might be carcinogenic", Washington, DC

April Invited speaker: National Cancer Institute Sponsored Workshop on Mucosal Immunosureillance, Inflammation and Cancer, "Inflammation and carcinogenesis", Rockville, MD

May Invited speaker: John Hopkins School of Medicine Jr. Faculty Research Program IBD Symposium, Management problems in IBD: Immunomodulators, Biologics and Dysplasia. Baltimore, MD

State of the Art Lecture and session co-chair: American Gastroenterological Association Annual Meeting, "Stem cells and cancer", Chicago, IL

Invited speaker: AGA Postgraduate Course, Digestive Diseases Week 2005, "Gastritis and cancer", Chicago, IL

Invited speaker: Japanese K-Club, Chicago, IL

June Invited Lecture: Hematology/Oncology Research Seminar, " ", New York University School of Medicine, New York, NY

Invited speaker and session chair: 10th US-Japan GI & Liver Meeting in the 21st Century, "Helicobacter and gastric cancer: a new paradigm for epithelial malignancy", Kyoto, Japan

Sept Invited Speaker: AGA-British Society of Gastroenterology (BSG) Meeting, "Stem cells and GI carcinogenesis", Oxford, England

Invited Lecture: GI Grand Rounds and Research Seminar, "Helicobacter, inflammation and stem cells: a new paradigm in epithelial cancer", University of Pennsylvania, Philadelphia, PA

Invited Speaker: World Congress of Gastroenterology, "H pylori, gastritis and gastric cancer: pathologic mechanisms", Montreal, Canada

Conferences Organized:

November 1999:	Third International Conference on Gastrin
September 2002:	International Regulatory Peptide Symposium
May 2004:	AGA/GRG Symposium: Stem cells and cancer stem cells.
May 2004:	AGA Spring Postgraduate Course (SPGC)
October 2004	FASEB conference: Gastrointestinal Response to Injury: Canada 2004
Planned for 2006:	AGA Symposium on "Stem cells in Cancer and Development"

NIH Study Sections:

Ad hoc:	Program Projects: 1994, 1996
	NIDDK-C 1999, 2000
Member:	NIDDK-C (6/2000-6/2004)
	NIH Gastrointestinal Cell & Molecular Biology DDK-GCMG (2004-present)

Patents filed:

2002 Diagnosis and treatment of gastrointestinal disease (Role of gastrin isoforms in the susceptibility to gastric atrophy and cancer). US Serial No. 10/257, Filed 10/8/02.

2003 Origins of gastric cancer (pending).

2003 Histamine and CCK2/gastrin receptor blockade in the treatment of acid-peptic disease and cancer (pending).

Bibliography

Original Peer-Reviewed Articles:

1. Linday L, Dobkin JF, Wang TC, Butler VP, Shaha JR, Linderbaum J. Digoxin inactivation by gut flora in infants and Children. Pediatrics. 1987; 79:544-548.
2. Parsonnet J, Welch K, Compton C, Strauss R, Wang TC, Kelsey P, Ferraro MJ. Simple microbiologic detection of campylobacter pylori. J Clin Micro. 1988; 26:948-949.
3. Brand SJ, Wang TC. Gastrin gene expression and regulation in rat islet cell lines. J Biol Chem. 1988; 263:16597-16603.
4. Green PHR, O'Toole KM, Slonim D, Wang TC, Weg A. Increasing incidence and excellent survival of patients with early gastric cancer: experience of a United States medical center. Am J Med. 1988; 85:658-661.
5. Lee EY, Wang TC, Clouse RE, DeSchryver-Kecskemeti K. Mucosal thickening adjacent to gastric malignancy: association with epidermal growth factor. Modern Pathology. 1989; 2:397-402.
6. Wang TC, Brand SJ. Islet cell specific regulatory domain in the gastrin promoter contains adjacent positive and negative DNA elements. J Biol Chem. 1990; 265:8908-8914.
7. Strauss RM, Wang TC, Kelsey PB, Compton CC, Ferraro MJ, Perez-Perez G, Parsonnet J, and Blaser MJ. Association of helicobacter pylori infection with dyspeptic symptoms in patients undergoing gastroduodenoscopy. Am J Med. 1990; 89:464-469.
8. Awad JA, Lee EY, Wang TC, Deschryver-Kecskemeti K, Clouse RE. Effect of mucosal thickening near gastric carcinoma on the endoscopic diagnosis of malignancy. Dig Dis Sci. 1990; 35:317-320.
9. Richter JM, Wang TC, Fawaz K, Bynum TE, Fallon D, and Shapleigh C. Practice patterns and costs of hospitalization for upper gastrointestinal hemorrhage. J Clin Gastro. 1991; 13:268-273.
10. Shiota G, Rhoads DB, Wang TC, Nakamura T, and Schmidt EV. Hepatocyte growth factor inhibits growth of hepatocellular carcinoma cells. Proc Natl Acad Sci USA. 1992; 89:373-377.
11. Wang TC, Brand SJ. Function and regulation of gastrin in transgenic mice: A review. Yale J. Biol. Med. 1992; 65:705-713.
12. Wang TC, Bonneir-Weir S, Oates P, Chulak M, Merlino GT, Schmidt EV, Brand SJ. Pancreatic gastrin stimulates islet differentiation of TGF-alpha-induced ductular precursor cells. J Clin Invest 1993; 92:1349-1356.
13. Tillotson LG, Wang TC, Brand SJ. Activation of gastrin transcription in pancreatic islet cells by a CACC promoter element and a 70 kDa sequence specific DNA binding protein. J Biol Chem January 1994, 269:2234-2240.
14. Jeffrey GP, Babyatsky M, Oates PS, Wang TC, Brand SJ. Spasmolytic polypeptide - an abundant trefoil peptide secreted luminally from mucous cells in adult and fetal rat stomach. Gastroenterology 1994 106:336-345.
15. Shiota G, Wang TC, Nakamura T, Schmidt EV. Hepatocyte growth factor in transgenic mice; effect on hepatocyte growth, liver regeneration, and gene expression. Hepatology, April, 1994, 19:962-972.

16. Wang TC, Cardiff RD, Zuckerberg L, Lees E, Arnold A, and Schmidt EV. Mammary hyperplasia and carcinoma in MMTV-Cyclin D1 transgenic mice. Nature June 23 1994; 369:669-671.
17. Sharp R, Babyatsky M, Takagi H, Tagerud S, Wang TC, Bockman DE, Brand SJ, and Merlino G. Transforming growth factor alpha can disrupt the normal program of cellular growth and differentiation in the gastric mucosa of transgenic mice. Development 121:149-161, 1995.
18. Wang TC, Babyatsky M, Oates PS, Zhang Z, Tillotson L, Chulak MB, Brand SJ, Schmidt EV. The rat gastrin-human gastrin chimeric transgene directs antral G cell specific expression in transgenic mice. Am. J. Phys. 268 (Gastrointest. Liver Physiol. 31): G1025-G1036, June, 1995.
19. Haase VH, Wang TC, Schmidt EV, and Bernards A. Normal lymphopoiesis in transgenic mice over-expressing the ltk transmembranous tyrosine kinase. Transgenics 1995, 1:487-495.
20. Koh TJ and Wang TC. Molecular cloning and sequencing of the murine gastrin gene. Biochem. Biophys. Res. Commun. 1995; 216:34-41.
21. Fox JG, Li X, Cahill R, Andrutis K, Rustgi AK, Odze R, and Wang TC. Hypertrophic gastropathy in Helicobacter felis infected wild type C57BL/6 mice and p53 hemizygous transgenic mice. Gastroenterology 1996 110:155-166.
22. Höcker M, Zhang Z, Fenstermacher DA, Tagerud S, Chulak MB, Joseph D, and Wang TC. The histidine decarboxylase promoter is regulated by gastrin through a protein kinase C pathway. Am. J. Physiol. 270 (Gastrointestin. Liver Physiol. 33):G619-G633, 1996.
23. Liang TJ, Reid AE, Xavier R, Cardiff RD, and Wang TC. Transgenic expression of tpr-met oncogene leads to development of mammary hyperplasia and tumors. J. Clin. Invest. 97:2872-2877, 1996.
24. Zhang Z, Höcker M, Koh TJ, and Wang TC. The human Histidine Decarboxylase promoter is regulated by gastrin and PMA through a downstream cis-acting element. J. Biol. Chem. 271:14188-14197, 1996.
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26. Höcker M, Koh TJ, Zhang Z, and Wang TC. Regulation of histidine decarboxylase gene expression. Yale J. Biol. Med. 1996; 69:21-33.
27. Höcker M, Zhang A, Merchant JL, and Wang TC. Gastrin regulates the human histidine decarboxylase promoter through an AP-1-dependent mechanism. Am. J. Physiol. 272 (Gastrointest. Liver Physiol. 35): G822-G830, 1997.
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29. Nakagawa H, Wang TC, Togawa K, Zukerberg L, Odze R, Wilson JB, and Rustgi AK. Overexpression of cyclin D1 in the esophageal epithelium results in dysplasia in transgenic mice. Oncogene 1997; 14:1185-1190.

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58. Colucci R, Fleming JV, and Wang TC. "L-histidine decarboxylase decreases its own transcription through downregulation of ERK activity". Am. J. Physiol. 2001;281:G1081-1091.
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66. Shea T, Wang TC, and Ferris T. A program to improve the management of patients on long term acid suppression. Journal of Clinical Outcome Management 2002; 9(6):312-318.
67. Kirton CM, Wang TC, Dockray GJ. Regulation of parietal cell migration by gastrin in the mouse. Am J Physiol Gastrointest Liver Physiol 2002 ; 283:G787-G793.
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EXHIBIT B

Documents Considered

Legal Pleadings

- Consolidated Amended Complaint for Violation of the Federal Securities Laws, dated October 27, 2003
- Plaintiffs' Memorandum of Law in Support of Plaintiffs' Motion for Class Certification, dated April 3, 2006

FDA Documents

- Transcript of the February 7, 2001 FDA Arthritis Advisory Committee meeting

CLASS Study Documents

- CLASS Study Protocols N49-98-22-035; N49-98-12-102
- A Plan Of The Final Analysis For Celecoxib Incidence Of Clinically Significant UGI Adverse Events vs. Ibuprofen & Diclofenac In OA Or RA (Studies N49-98-22-035 and N49-98-12-102), dated October 7, 1999

All other sources cited in report and exhibits.